available evidence to support intervention with more frequent assessment or by different healthcare professionals is limited in quantity and quality.

Purists may argue that screening for risk in early pregnancy and recommendations for the proposed frequency of antenatal screening visits does not meet all the criteria for screening. However, in 46% of maternal deaths and 65% of fetal deaths reported via the confidential inquiries into maternal deaths,[1] different management would reasonably have been expected to alter the outcome. Many of these management problems arise in the community because of a failure to identify and act on established risk factors at booking and to recognise and respond to signs and symptoms consistent with pre-eclampsia. We may not have the answers in terms of the outcome from such intervention, but we cannot be complacent in the face of the recurrent deficiencies identified in the confidential inquiries. The pragmatic approach of PRECOG is essential because pre-eclampsia matters.

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Counting the dead in Iraq

We need to know how many people have died

Counting the dead is intrinsic to civilised society. Understanding the causes of death is a core public health responsibility. The government's white paper on public health emphasises the vital role of assessing the impact on health of all public policy.[1] This is well recognised, and yet neither the public nor public health professionals are able to obtain reliable and officially endorsed information about the extent of civilian deaths attributable to the allied invasion of Iraq. Estimates vary between tens and hundreds of thousands.

These estimates come from reports in the press, or counting bodies admitted to hospitals, (www.iraqbodycount.net) as well as surveys. The former are likely to be inaccurate and to underestimate the true numbers and do not easily allow for reliable attribution between, for example, violent and natural causes. Public access to reliable data on mortality is important. The policy being assessed—the allied invasion of Iraq—was justified largely on grounds of democratic supremacy. Voters in the countries that initiated the war, and others—not least in Iraq itself—are denied a reliable evaluation of a key indicator of the success of that policy. This is unacceptable.

Instead the UK government's policy was first not to count at all, and then to rely publicly on extremely limited data available from the Iraqi Ministry of Health. This follows US government policy; famously encapsulated by General Tommy Franks of the US Central Command "We don't do body counts."[2] Its inadequacy was emphasised after the publication of a representative household survey that estimated 100 000 excess deaths since the 2003 invasion.[3] The government rejected this survey and its estimates as unreliable; in part absurdly because statistical extrapolation from samples was thought invalid.[4] Imprecise they are, but to a known extent. These are unique estimates from a dispassionate survey conducted in the most dangerous of epidemiological conditions. Hence the estimates, as far as they can go, are unlikely to be biased, even allowing for the reinstatement of Falluja. To confuse imprecision with bias is unjustified.

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The methods for counting the dead in such circumstances are well established and cannot rely on incidental reports or assessments in hospital mortuaries alone. They require first hand verbal autopsies,[5] which should be reliably obtained so that extrapolation to the population is possible, as Roberts et al had done. They also require some linkage with unclassified data on military offensives.[6] Although active surveillance of this kind is extremely difficult in the context of such violence, even limited household surveys are essential so long as they are systematic. Such data can then be combined with information from passive sources to establish a more accurate overall assessment.

Counting casualties accurately can help to save lives both currently and in the future. Understanding the burden of death, injury, disease, and trauma that the population is currently suffering enables proper planning of war, and health, and in assessing local responses appropriately. In the future this should help government and military planners to assess the likely humanitarian implications of conflict.

The plain fact is that an estimate of 100 000 excess deaths attributable to the invasion of Iraq is alarming. This is already half the death toll of Hiroshima.[7] Apart from the practical arguments, the principled ones stand and will always stand. Have we not learnt any lessons from the history of sweeping alarming numbers of deaths under the carpet? This is not something about which there can be any political discretion 60 years after Auschwitz. The UK government, acting on our behalf, ought to offer reasoned criticism of the existing estimates. It should pursue their public health responsibilities to count the casualties by using modern methods. Democracy requires this, as does proper responsibility under the Geneva Conventions.

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The sources the government prefers are likely to be seriously biased for several reasons. They do not take into account deaths during the first 12 months since the invasion[8]; only violence related deaths reported through the health system are taken into account (very likely to lead to an underestimate[9]); and non-violent deaths due to the destruction of war are not taken into account. Furthermore, even these limited figures are no longer being released on request.[10]

Apparently the defence ministry has set up an appraisal group,[11] but we urgently await transparency and public accountability. The time elapsed since the announcement is already longer than it took to conduct the field survey last year. Electorates, in Iraq and elsewhere, have a right to know. To procrastinate further for no good reason is to devalue public health processes, not to mention Iraqi lives. As public health professionals we need to know the health costs.

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Diabetes, insulin therapy, and colorectal cancer

Evidence indicates a modest increase in risk of bowel cancer among people with type 2 diabetes

For two decades, investigators have recognised the overlapping risk factors for type 2 diabetes mellitus and colorectal cancer—obesity, Western diet, and sedentary lifestyle—and speculated about a link between these two common diseases. Accumulating evidence shows that type 2 diabetes mellitus is associated with a 40-60% increased risk of cancer of the large bowel,[1 2] and specifically, proximal colonic malignancy.[3] These associations are independent of body mass index and are more consistently reported than those with breast and endometrial cancers. Recent data from the European Prospective Investigation into Cancer (EPIC-Norfolk) study show that this increased risk is largely explained by changes in glycated haemoglobin (HbA_{1c}) concentrations.[4] This implies that glycaemic control is likely to be important in determining which patients develop colorectal cancer. In contrast to type 2 diabetes mellitus, no associations have been found between colorectal cancer risk and type 1 diabetes mellitus, nor gestational diabetes.[5]

Recently published data from the large US Cancer Prevention Study II (1.2 million men and women) confirmed findings from previous small studies that the presence of diabetes may influence outcome in patients with malignancy of the large bowel.[6] Furthermore, in the setting of a large randomised controlled trial of adjuvant chemotherapy of stages II and III colon cancer, Meyerhardt et al reported among people with diabetes mellitus (categorisation into types was not reported) significantly higher rates of overall mortality and reduced disease free and recurrence free survivals—even after other predictors of outcome had been adjusted for.[7] Importantly, this study showed that the disease free and overall survival curves in the first two years were almost identical for patients with and without diabetes. Although these observations need to be replicated, they imply that among people with diabetes who have colorectal cancer, some unidentified mechanism may influence progression of disease unfavourably some time after initial treatment, rather than the perception that diabetes is associated with advanced presentation, compromised initial treatment, and increased early postoperative mortality.

The effects of diabetes mellitus on colorectal cancer may be mediated through mechanisms ranging from increased colonic transit time to hyperinsulinaemia. In relation to the latter, at least in the early phase of development, type 2 diabetes mellitus is associated with increased circulating insulin concentrations. Insulin may stimulate cell proliferation through two pathways: a minor pathway that entails direct activation of the insulin receptor or insulin-like growth factor (IGF)-I receptor, and a major pathway via inhibition of IGF binding proteins (in particular, IGFBP-1 and IGFBP-2), resulting theoretically in increased bioavailability of IGF-I to the IGF-I receptor. An important role for IGF-I in colorectal carcinogenesis is supported by epidemiological studies and animal models.[8 9] Clinical studies also independently link high circulating concentrations of C-peptide, as a marker of insulin production, with increased colorectal cancer risk.[10]

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